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Bronchiectasis.—Bronchiectasis was first described in 1819 by Laennec. One hundred years later the use of lipiodol in conjunction with radiography made possible the exact demonstration of bronchial dilatation. During recent years, the treatment and diagnosis of bronchiectasis have been improved by the perfection of the bronchoscope and bronchoscopic lavage. The clinical picture is well defined. The disease usually begins in early childhood and dates from an acute respiratory illness. The history, long duration of the illness, the paroxysmal cough, copious sputum, and the variable physical signs in the chest form a clinical syndrome which is unmistakable. Bronchiectasis may be confused with tuberculosis, chronic bronchitis and chronic cough produced by nasopharyngeal catarrh. Unilateral bronchiectasis with massive fibrosis has a relatively benign course, probably due to the partial collapse of the lung resulting from fibroid contraction and to the absence of great bronchial dilatation and catarrh. Bilateral bronchiectasis is more common. Less fibrosis with greater dilatation of the bronchioles, more copious sputum and greater impairment of health are observed. It is very difficult to give an approximate estimate of the frequency of the disease, but it may be said that the condition, in its established form, is uncommon. The cure of bronchiectasis seems improbable but cases are on record in which great and apparently permanent amelioration of symptoms have occurred. In postmortem studies of 5 early cases, it was found that the primary changes are ulceration, suppuration, and destruction of the bronchial walls. These walls are replaced by granulation tissue and by cubical and nonciliated epithelium. In these areas the lumina of the bronchi are converted into irregular spaces. Adjoining these areas young fibrous tissue infiltrates the lung. Treatment consists of postural drainage, general hygienic measures, collapse of the lung in unilateral cases, and bronchoscopic lavage. The more drastic measures which extirpate the affected area are attended with a high mortality and the results are not encouraging.—*Remarks on Bronchiectasis, C. McNeil, Brit. M. J., August 6, 1932, no. 3735, 229.*—(*M. E. P.*)

Bacteriology of Bronchiectasis.—This report is based on an analysis of materials made available through the operative procedure of lobectomy in 9 consecutive cases of bronchiectasis. Every support was afforded for making bacteriological analysis immediately on the removal of the lobe of the lung. The bronchi were opened under aseptic precautions. Smears and dark-field preparations were then made from the contents at various points, care being taken to select granular material, if present, and also from scrapings of the bronchial mucosa, in order to pick up organisms that had penetrated it. With similar material, cultures were made aerobically and anaerobically on blood and "chocolate" agar, and in broth, cooked meat and Rosenow's deep brain-broth medium. When indicated, sub-

cultures were made from the various media, and identification of the organisms was completed. No attempt was made to grow spirochaetes, their presence or absence being determined by dark-field examination and by smears stained by Fontana's method. The bacteriological procedure was usually completed half an hour after removal of the diseased lobe, and the tissues were further studied histologically. In only 4 of the 9 cases was it possible to demonstrate spirochaetes by dark-field illumination, and in two of these they were seen in tissue sections stained by Levaditi's method. Fusiform bacilli likewise were present on 5 occasions. In only one of the cases were the spirochaetes numerous. A wide variety of microörganisms has been cultivated in these cases with variations in the duration of the disease from three months to thirteen years. These results, obtained under favorable conditions, certainly suggest that no one microörganism and no combination of microörganisms can be held responsible for the damage to the bronchial wall. In this series streptococci of different types predominated, but, since these organisms are the commonest invaders in many infectious processes, too much stress cannot be laid on this finding. Spirochaetes could not be demonstrated in the lungs in many cases of bronchiectasis, and their aetiological relationship to the disease is to be questioned. Their presence in the lesions may favor chronicity, but they probably have no greater aetiological significance than have other bacteria.—*The Bacteriology of Bronchiectasis: an Analysis Based on Nine Cases in Which Lobectomy Was Done, P. H. Greey, J. Infect. Dis., March, 1932, l, 203.*—(*F. G. P.*)

Phrenicectomy for Bronchiectasis.—A number of authors have reported very satisfactory clinical results in the treatment of bronchiectasis by phrenicectomy. Three cases are here reported in which the efficacy of this procedure is confirmed. The diagnosis in each case was corroborated by roentgenograms taken after the intratracheal injection of lipiodol. Each had been refractory to medical methods. In two cases the lesion was unilateral and the duration of symptoms prior to operation had been 9 years and 12 years. In both an ascent of the diaphragm of three finger-breadths was demonstrated. Relief of symptoms was marked in one month and complete in 14 and 20 months respectively. Roentgenograms showed concomitant anatomical cure in the latter case, while no anatomical changes whatever had taken place in the former case. The third case presented a bilateral lesion of two years' duration. Phrenicectomy was performed on the side with the most marked involvement. Although the ascent of the diaphragm was slight, marked amelioration of symptoms was apparent in six months. The amount of sputum had decreased by three-fourths, and a satisfactory gain of weight and appetite had occurred. Again no anatomical regression of the lesion could be demonstrated roentgenologically. A contralateral phrenicectomy is being considered. Phrenicectomy sometimes yields only transitory relief; hence the permanence of these results is still questionable. Occasionally it is totally inefficacious. The degree of perifocal sclerosis and pleural symphysis is probably the determining factor. Despite the possibility of failure it is urged that phrenicectomy is the operation of choice in bronchiectasis in preference to artificial pneumothorax. The latter is often unsuccessful and incomplete because of pleural adhesions and also imposes the added burden on the patient of reinsufflations. The mode of action of phrenicectomy is somewhat obscure especially as in two of these cases the lesion remained unaltered.—*Dilatation des bronches et phrénicectomie, A. Courcoux, A. Bidermanli and A. Alibert, Rev. d. l. Tuberc., April, 1932, xiii, 401.*—(*A. E. M.*)

Spontaneous Pneumothorax.—During two and one-half years, 368 patients were treated with artificial pneumothorax, and during this period 13 patients showed spontaneous pneumothorax sixteen times. In 3 patients it occurred immediately after the institution of the pneumothorax. In these cases the visceral pleura was probably injured by the needle. Four other patients reported that they had the feeling of something tearing in the chest at different intervals after their inflation. It may be assumed that a tearing of adhesions took place. In 6 other patients pneumothorax was probably due to a rupture of emphysematous blebs. For the treatment of patients with spontaneous pneumothorax with increasing intrapleural pressure, the introduction of a permanent cannula is advised, and it is pointed out that morphine and cardiac stimulants are indicated.—*Über ein und doppelseitigen Spontanpneumothorax, Stein and Boedeker, Beitr. z. Klin. d. Tuberk., August, 1932, lxxx, 415.*—(*M. P.*)

Rupture of Lung.—A girl, 15 years old, was treated for pulmonary tuberculosis with bilateral artificial pneumothorax and phrenicectomy on the left side. Five days after the first inflation on the right side, a spontaneous valvular pneumothorax developed on this side. There were critical

symptoms of high positive pressure, but with repeated decompressions, as indicated by the symptoms, the danger was overcome, and treatment could be continued in the regular way.—*Ein Beitrag zum Verlaufe und zur Behandlung eines röntgenologisch beobachteten einseitigen spontanen Ventilpneumothorax mit günstigen Ausgange als Komplikation bei doppelseitigem künstlichem Pneumothorax und kombinierter einseitiger künstlicher Phrenicusexairese im Kindesalter, K. Nüssel, Beitr. z. Klin. d. Tuberk., June, 1932, lxxx, 158.*—(*M. P.*)

Bilateral Spontaneous Pneumothorax.—A case of benign bilateral pneumothorax with recovery is reported, the rupture of the second lung taking place before the complete reëxpansion of the first. The patient was a white male aged 27 years, who complained of cough and a dull pain over the left anterior midchest, which was aggravated on movement, but not on deep breathing. Roentgen-ray examination revealed a pneumothorax of the left hemithorax. About six weeks after the diagnosis had been established, he noticed he was unusually dyspnoeic and complained of a feeling of fulness in the chest. These symptoms appeared without apparent cause. This called for fluoroscopic examination, which showed the right lung to be about 80 per cent collapsed, and a small amount of fluid in the sulcus. Spontaneous bilateral pneumothorax is mostly a sequel of evolutive pulmonary tuberculosis. About 58 per cent of the cases are of this type. The absence of evidence of clinical pulmonary tuberculosis on physical and roentgen-ray examinations, together with failure to demonstrate tubercle bacilli in the sputum or pleural fluid on smear, culture or animal inoculation, rules out tuberculosis as an aetiological factor in this case.—*Spontaneous Bilateral Pneumothorax. With Report of a Case and Review of Literature, B. T. McMahon, Am. J. M. Sc., May, 1932, clxxxiii, 695.*—(*W. I. W.*)

Recurring Spontaneous Pneumothorax.—A man, aged 21, had a small haemoptysis twelve months after the onset of tuberculosis. Shortly afterward he developed a spontaneous pneumothorax on the left, while at bed-rest. He remained in bed six months, and was then admitted to a sanatorium with his lung fully reëxpanded, but with active disease in both lungs. Six weeks later, while at bed-rest, he developed a spontaneous pneumothorax on the left. Dyspnoea became severe and a needle was allowed to remain in the chest-wall so that air could escape beneath fluid in a bottle. It was necessary to keep the needle in place ten days. A roentgenogram eight weeks later showed the lung half reëxpanded. No pleural exudate was demonstrated on the occasion of either pneumothorax.—*A Case of Recurring Spontaneous Pneumothorax with Acute Pressure Symptoms, H. Selby, Lancet, January 30, 1932, ccxxii, 238.*—(*R. M. F.*)

Pulmonary Gas Absorption in Bronchial Obstruction.—Two new methods are described which allow a physiological study of the gas exchanges in a lobe or a whole lung after its bronchus has been completely obstructed. With both methods complete bronchial obstruction is obtained by a special type of cannula, through which gas samples may be drawn from below the obstructed portion of the lung. With the first method, the pulmonary changes are followed by roentgen-ray and fluoroscopy, the chest being intact. By the second technique, the chest is wide open, allowing direct observation of the lungs through an "oscillating negative pressure box," which closely simulates the physiological condition of a closed thorax.—*Studies in Pulmonary Gas Absorption In Bronchial Obstruction. Two New Methods for Direct and Indirect Observation, P. N. Coryllos and G. L. Birnbaum, Am. J. M. Sc., March, 1932, clxxxiii, 317.*—(*W. I. W.*)

Aerodynamics of Bronchial Obstruction.—Certain observations made experimentally on dogs are reported. By means of an apparatus which included (1) two cannulas, one of which had an expansile cone-shaped tip capable of obstructing a bronchus, the other opening proximally to the obstruction, (2) a connecting U-tube with a valve interposed, and (3) manometers connected proximally and distally to the valve, it was possible to observe the pressure changes occurring in the bronchi during respiration and during cough, and also to make observations involving either lobar or lobular obstruction. *Conclusions:* In all varieties of bronchial obstruction the intrabronchial pressure distal to the point of obstruction oscillates with respiration at about the level of atmospheric pressure, rising above it in expiration and falling below it in inspiration. The intrabronchial pressure proximal to the obstruction oscillates in a similar manner but much less widely. In *total lobar obstruction* the zone of oscillation of the distal intrabronchial pressure changes only as a result of absorption of the air. In *simple inspiratory lobar obstruction* any tidal air that may be included in the obstructed part of the lung to begin with escapes

quickly past the valve, but after this the pressure remains stationary and the obstructed part is not collapsed by the valve action. In *simple expiratory lobar obstruction* air enters rapidly through the valve until the obstructed part has reached its capacity for air, and then only a little enters from time to time. The thoracic parietes expand automatically to accommodate the inflated parenchyma. If the animal is killed the lung is found to be emphysematous. In the three types of *lobular obstruction* collateral respiration saves the obstructed part of the lung from such alterations of inflation as develop in lobar obstruction. If the obstruction is valvular, air enters or leaves by the valve for indefinite periods, and escape or supply of air by collateral connections proceeds concurrently. Neither atelectasis nor emphysema develops. In general, the above principles apply to cough. The displacing effect of cough depends on whether or not tidal air is contained in the obstructed part at the onset of the act. In the absence of tidal air the effect may be toward the alveoli; with adequate tidal air the effect is toward the trachea. Lobular obstruction undoubtedly occurs frequently. In bronchial catarrh a drop of moisture in a capillary respiratory duct often prevents the passing of air. When cough takes place at once the tidal air originally imprisoned serves for the elimination of the obstruction, but when the subject sleeps absorption proceeds and collateral respiration is necessary to compensate for the tidal air. A bronchial tree without provision for collateral respiration would be as inefficient as a blood-vascular system without collateral circulation, perhaps more so since bronchial obstruction is probably more common than arterial obstruction. Collateral respiration depends on the fact that the lobular divisions of the bronchial tree of any one pulmonary lobe are interconnected abundantly at the periphery, rather than being independent as has been generally supposed. The interlobular septa of the lung are incomplete, and the alveoli at the planes of fusion communicate with each other by minute passages, and perhaps, too, by diffusion through the alveolar walls.—*The Aerodynamics of Bronchial Obstruction, G. E. Lindskog and C. M. Van Allen, Arch. Surg., February, 1932, xxiv, 204.*—(*R. D.*)

Bronchiolitis Obliterans.—This condition, involving the terminal bronchi, has been reported as occurring as a sequel of infectious pneumonia and of pneumonia caused by chemical irritants. Three cases are here reported in which obliterative bronchiolitis was observed at the postmortem examination of patients dying of uraemia in Bright's disease. The condition is attributed to the disturbance of metabolism which occurs in Bright's disease, and is manifested by an increased tendency to exudation and decreased tendency to absorption. In two cases, at least, the bronchiolitis was caused by bacterial infection. Whether it can arise as the result of intoxication alone, as does general serositis, could not be decided.—*The Pathogenesis of Bronchiolitis Obliterans, W. Ehrich and J. F. McIntosh, Arch. Path., January, 1932, xiii, 69.*—(*R. D.*)

Lobar Pneumonia.—A summary of 27 patients suffering with early lobar pneumonia and treated with inhalations of 5 per cent carbon dioxide and 95 per cent oxygen is given. In this study there were 2 deaths. There were 22 male and 5 female patients. The age varied between 14 and 46 years. The average duration of illness before admission to the hospital was 2 days. The site of the pneumonia in the involved lung was as follows: right upper lobe, 7 cases; right middle lobe, 7 cases; right lower lobe, 10 cases; left upper lobe, 7 cases; left lower lobe, 10 cases. There was one case of pneumonia of the right hilum. The pneumonia spread to one or more lobes in 9 patients of the series. The gas was given on an average of 5½ days. The shortest period of administration was 3½ days, the longest 10½ days. The disease ended by crisis in 24 patients, and by lysis in 3. Sputum was typed in each case, with the following results: Type I, 1 case; type II, 2 cases; type III, 4 cases; type IV, 11 cases; mixed, 2 cases; undetermined, 7 cases. There were 15 positive blood-cultures. The pneumococcus was recovered in 7 cases, the streptococcus in 2 cases, the staphylococcus in 3 cases, and the diphtheroid bacillus in 3 cases. Determinations of the vital capacity served as a valuable guide as to the degree of consolidation and as an index of spread to other lobes. There was no constant response of the leucocytes before, during, or after inhalations. The number was increased at times, while at other times it was decreased during inhalations. The most common sensation experienced while the patients were inhaling the gas was headache. Seven patients complained of moderate to severe headache. Pleural pain was experienced by nearly all of the patients at the beginning of each inhalation but was not severe enough to be a contraindication for its use. Four patients complained of severe pleural pain. The majority of patients experienced some fatigue during inhalations, and when this

was severe the periods of administration were shortened, much to the comfort of the patient. One patient had severe cough as a result of inhaling the gas. Administration was stopped because in this patient subsequent empyema developed. Sixteen patients took the gas well and experienced considerable relief from symptoms through its use.—*Treatment of Lobar Pneumonia with Carbon Dioxide and Oxygen, Report of Twenty-seven Cases, J. F. Hanson and A. W. Calhoun, Arch. Int. Med., August, 1932, l, 269.*—(*W. I. W.*)

Pneumonic and Bronchopneumonic Residua.—The author reports 6 cases in which were found, particularly in the right upper lobe, infiltrations with extraordinarily slight or normal physical findings. Temperature and blood-sedimentation were normal, while the shadows were fairly extensive, homogeneous and sharply demarcated. Later they became more flocculent. There was negative sputum as well as a favorable course, especially from the roentgenological standpoint. The shadows would disappear in a few weeks and residual scars were not found. The cases are considered to be residua following pneumonic and bronchopneumonic processes. The danger of diagnosing these cases as tuberculosis is pointed out.—*Pneumonische und bronchopneumonische Restzustände und ihre differentialdiagnostische Bedeutung, F. Kellner, Beitr. z. Klin. d. Tuberk., 1931, lxxvi, 523.*—(*H. J. C.*)

Pneumonia Due to Aspirated Oils in Infants.—Two cases of infants aged four months, who were ill for a long time, and finally died of extrapulmonary infection are reported. During their illness the pulmonary signs and symptoms did not dominate the clinical picture, and all tests for tuberculosis were consistently negative. Each child was given oil over a considerable period of time, through the nasopharynx. The roentgenological findings presented a very accurate picture of the gross pathological changes in the lungs, and they indicated much more extensive pulmonary involvement than was brought out by the clinical examination. They revealed hazy indefinite shadows of irregular density scattered throughout both lungs. The pathological reports revealed that there were large quantities of fatty material in the lungs, and that there was evidence of chronic pneumonia and fibrosis. The widespread use of oils in pediatrics creates a large potential source for this condition.—*Some Unusual Pneumonias Associated with the Aspiration of Fats and Oils in the Lungs, J. W. Pierson, Am. J. Roentgenol. and Rad. Ther., April, 1932, xxvii, 572.*—(*W. I. W.*)

Artificial Pneumothorax in Lobar Pneumonia.—Six cases of acute lobar pneumonia treated by artificial pneumothorax are described. The patients' ages ranged from 9 to 55 years. Pneumothorax was induced in 3 of the young adults on the third day of disease, and in the other young adult and the child on the fifth day of the disease. In all of these cases an "artificial crisis" occurred immediately. This consisted of profuse perspiration, decrease in cyanosis and dyspnoea, disappearance of thoracic pain, and drop in temperature, and in respiratory and pulse-rate. In the cases of the child and two of the young adults one refill each was given before convalescence was established. It was found that the acute symptoms recurred if the air was allowed to be absorbed, and that this occurred in approximately 24 hours. Then a refill produced a second "crisis." Another young adult was given one refill, but the amounts of air were smaller. His symptoms were definitely decreased, but more time was required to establish convalescence. Later a partially healed tuberculous lesion was discovered in the opposite lung, and tubercle bacilli were found in the sputum. Resolution of the pneumonia progressed satisfactorily. The other resisted treatment and received only small amounts of air upon two occasions. His symptoms decreased but recurred, and continued until the normal crisis on the seventh day. The patient fifty-five years of age had been ill several days and on admission was cyanotic, with mild fever and a pulse of poor quality. An unusually large amount of air was introduced and, though a "crisis" occurred with temporary improvement, the patient died soon afterward of failure of the right ventricle. Postmortem examination was not made. To identify the "artificial crisis" with the natural one, estimates of the urinary chlorides and the leucocytes were made in one case before and after the defervescence produced by the pneumothorax. There was an increase of 60 per cent in the urinary chlorides and a fall in the leucocyte count of over 25 per cent in less than 24 hours after the induction of pneumothorax. Points emphasized are as follows: (1) induction of pneumothorax initiates immediately a series of events identical with the "crisis" of pneumonia; (2) the pleura is hyperalgesic, and absorbs air more rapidly than normally; and (3) maintenance of pneumothorax for approximately 48 hours initiates convalescence, and resolution oc-

curs with less damage to the lung than in untreated cases. Certain dangers, such as pyopneumothorax and cardiac failure, are discussed. There was no pleural effusion in the cases described. Small, frequent refills are suggested for elderly persons, in order to avoid cardiac collapse. No particular site for injection of air is preferred as the pleura is uniformly hyperalgesic. A procedure suggested is as follows: (1) preliminary morphine; (2) local anaesthesia; (3) asepsis; (4) induction of 400 to 600 cc. of air during the inspiratory phase; (5) a refill of 300 to 500 cc. after 12 hours; (6) another refill of 100 cc. 12 hours later, if the disease is not controlled; and (7) the use of diaphoretics and Felton's serum as indicated.—*Treatment of Acute Lobar Pneumonia by Artificial Pneumothorax, J. J. Coghlan, Lancet, January 2, 1932, ccxxii, 13.*—(*R. M. F.*)

Acute Pulmonary Oedema.—Acute pulmonary oedema is defined as a suffocation crisis, which is accompanied by a more or less abundant, frothy, often red, and always strongly albuminous expectoration, and which is due to a sudden flooding of the pulmonary alveoli by a profuse serous transudation. The most important aetiological factors are as follows: Syphilitic aortitis with insufficiency of the aortic valve is the most frequent cause. Other forms of aortitis, acute and chronic, including aneurysms and rheumatic valvular disease, may be factors. Of second importance is chronic hypertension of a moderate or marked degree, either with or without nephritis. Acute nephritis with normal blood-pressure may result in acute pulmonary oedema. A number of theories have been advanced concerning the pathogenesis involved, but the one now accepted is the dualistic, cardiovasomotor theory of Merklen and Lian, which considers acute pulmonary oedema as a form of paroxysmal left ventricular insufficiency, the other forms are cardiac pseudoasthma and angina pectoris. These three forms frequently alternate in the same individual, while evidence of chronic progressive left ventricular insufficiency, such as dyspnoea, murmurs, and a rapid or alternating pulse, frequently exist following an attack of pulmonary oedema. It has been proved that, in addition to the cardiac factor, a vasomotor sympathetic nervous factor is essential. There seems to be either an active capillaro-arterial vasodilatation of the pulmonary circulation, or an active capillarovenous constriction of the systematic circulation, which causes distention of the pulmonary vessels and surcharging of the left heart. The heart fails to cope with the added load and the result is a passive congestion of the lung. Of the numerous forms recognized clinically, the most important ones are as follows: In aortitis and hypertension there is a bronchoplegic, fulminating type which is fatal before formation of sputum. In mitral stenosis there may be a severe type; but usually there is a mild, discrete "crisis," a few coughs with slight transient dyspnoea and a small amount of rosy mucoid sputum. It is usually precipitated by the labor of pregnancy, strong emotion, intense effort, or an infectious disease. In acute infectious diseases there may be the mild type that follows acute respiratory infections in a hypertensive individual, the severe type that occurs in the course of acute articular rheumatism or "la grippe" without preliminary pulmonary involvement, and that associated with pneumococcic or typhoid infections. Fulminating and slow types occur secondary to inhalation of asphyxiating gases. The localized form found in subacute infectious oedemata and in cardiac insufficiency is really an oedematous passive congestion. In the typical clinical picture, onset is in the middle of the night with coughing and dyspnoea of increasing severity, followed by an abundant, frothy, rose- or salmon-colored expectoration. Fine râles appear at the base of the lung and increase until heard throughout. With early therapy there is usually a rapid recovery. Death may occur from asphyxiation or from cardiac failure. Prognosis depends upon the ease and abundance of expectoration, the resistance of the cardiovascular system, and upon how soon therapy is instituted. Abundant expectoration is a good sign, indicating relief of the congestion. Since there are usually recurrences, some member of the patient's household should be taught how to use a cupping glass so that the patient can get some relief while waiting for a doctor. Therapy consists essentially in the withdrawal of 500 to 800 gm. of blood from a vein. One centigram of morphine chloride exerts a beneficial cardiodynamic and antispasmodic effect, but is contraindicated in the presence of a severe nephritis. One fourth milligram of ouabain should be given intravenously daily for three to five days, and the patient kept at complete rest. Theobromine, digitalis, luminal or other sedatives, reduction of liquids in the diet and dechloruration are of value in prolonging the interval between attacks. Adrenalin should be avoided in one predisposed to acute pulmonary oedema.—*L'oedème pulmonaire aigu, C. Lian, Arch. Med.—Chirurg. d. l' Appareil Resp., 1931, vi, 345.*—(*E. K.*)

Friedländer-Bacillus Pulmonary Infection.—An extremely acute type of Friedländer-bacillus infection is reported, the patient living only twenty-six hours after onset. The clinical findings corresponded to those usually reported for the disease. The pathological findings were those of an uncommon lobar pneumonia instead of a confluent bronchopneumonia, or a "pseudo" lobar pneumonia. The disease began by a rapid growth of the encapsulated microörganisms in the alveoli and smaller bronchi, causing an exudate rich in oedema fluid containing scattering monocytes, irregularly placed fibrin, with occasional haemorrhages into the alveoli. This aspect has only a gray to yellow-gray appearance grossly, and accounts for the infrequent appearance of the red stage of hepatization. Death occurred before the other stages could develop. Ordinarily the evolution of the lesion from the "red" stage is characterized by an infiltration of polymorphs along the alveolar walls, forming in crescents along the plugs composed of bacilli, monocytes, and varying amounts of fibrin. Later an invasion of the alveolar wall results in a huge dilatation of the alveolar capillaries, followed by thrombosis, necrosis, and abscess-formation. The last two processes correspond to the stage of resolution of pneumococcus pneumonia. The dilatation of the alveolar capillaries, with an occasional rupture into the alveoli, gives to the lesions a mottled dark red appearance that is occasionally present. If the patient survives this stage, a gradual change to a chronic form ensues, with a substitution of lymphocytes and plasma cells for the polymorphs and an invasion of the older lesions with fibroblasts and connective tissue, and a true metaplasia forming squamous or cuboidal epithelial cells over the cavity walls. When the disease is progressive, there is a continuous sequence of lesions that begins in small bronchopneumonic foci and passes through the various stages of evolution described above, usually extending downward toward the base until death.—*Pulmonary Infections by the Friedländer's Bacillus (Bacillus Mucosus Capsulatus), F. Fremmel, K. J. Henrichsen and H. C. Sweany, Ann. Int. Med., January, 1932, v, 886.*—*(R. M. F.)*

Psittacosis.—Five cases of psittacosis occurring in one family are presented with autopsy observations in one. The disease in these cases is attributable to exposure to infected love-birds or parakeets brought to this country from Havana. Biological evidence of the identity of the disease was possible in this epidemic; the virus of psittacosis was demonstrated in the sputum of one of the patients; in the liver, lung, and spleen of another patient; and in the liver and spleen of the only bird available for examination. The characteristic clinical points of diagnostic importance in this group of cases were: (1) history of exposure to a diseased bird; (2) incubation period of approximately nine days; (3) marked headache; (4) toxemia, which was out of proportion to the physical findings in the chest; (5) slight cough with extremely scanty, thick, tenacious expectoration; (6) an extremely fine type of crepitant râles; and (7) roentgen evidence of dense consolidation of part of a lobe of the lung almost from the very onset of symptoms. One prominent feature of the disease was the tendency to thrombosis. There was no change in the bleeding or coagulation time in any case. Thrombi were found postmortem in the pulmonary vessels, right iliac vein and splenic vein in case 2. In case 3 a pulmonary embolus was the first indication of an extensive thrombosis of the veins of the left leg.—*Psittacosis: Report of Five Cases, M. A. Rabinowitz and S. H. Livingston, Arch. Int. Med., March, 1932, xlix, 464.*—*(W. I. W.)*

Pulmonary Lesions in Tularaemia.—Among 13 cases of tularaemia observed by the author 6 gave clinical evidence of intrathoracic disease, pleural effusion occurring twice, bronchopneumonia twice, and bronchitis and lung abscess once each. Autopsy reports gathered from the literature and covering 8 cases of tularaemia showed some abnormality of lungs or pleura in each. There is a similarity between the caseous necrosis of tularaemia and the lesions of tuberculosis. Bronchopneumonia, pleural effusion, and lung cavitation occur in both conditions. In the case reported infection followed the skinning of a dead opossum. The diagnosis of tularaemia was established by serological studies. Admission to the hospital followed the development of a cough with abundant, foul, bloody sputum. An X-ray showed areas of rarefaction in the lower half of the right chest, the largest being 3 x 6 cm. On the left the hilum shadows were heavy. A diagnosis of lung abscess on the right and probable bronchiectasis on the left was made. At autopsy the pleural cavities contained no fluid. The right lung was adherent at the base, and in this area a cavity 8 x 12 cm. with dirty gray necrotic walls was found. The pulmonary artery in this lobe was thrombosed. Thrombi were also found in the pulmonary arteries in the left lung, and consolidation was present in the area supplied. Microscopical studies

of these areas showed an accumulation of mononuclear cells and a proliferation of fibroblasts with a few polynuclears. The more necrotic areas suggested a caseous pneumonia, with mononuclears outlined in the exudate and pus absent.—*Pulmonary Lesions in Human Tularemia, S. D. Blackford, Ann. Int. Med., May, 1932, v, 1421.*—(*R. D.*)

Lung Abscess.—A case is reported which is believed to have been one of pneumonia of the right middle lobe with subsequent abscess formation. The various diagnoses made were malignant tumor, encapsulated empyema, lung abscess, and resolving pneumonia. There was a history of adequately treated syphilis, and the blood had been negative to the Kahn test for one year. The patient was hospitalized, when the third attack of pain in the right chest occurred within three months. He had had fever, night-sweats, and morning cough. There was an area of dulness, with absence of breath-sounds, corresponding to the middle lobe. A roentgenogram revealed a homogeneous density of the middle lobe. Five days after admission the patient expectorated copious amounts of pus, which had no odor, was negative for tubercle bacilli, and did not cause tuberculosis when inoculated into a guinea pig at a later date. The temperature became normal with the evacuation of the pus, and remained so. There was a history of similar expectoration of pus with relief of symptoms one month before hospitalization. The morning after expectoration of the pus began an attempt was made to aspirate the dense area of the middle lobe, without success. Expectoration continued and the classical signs of cavity were found over the middle lobe. Clearing and decrease in size of the density were seen by roentgenograms, and on the twenty-sixth day in the hospital it was seen that the dense area had resolved almost completely.—*Post-Pneumonic Lung Abscess Resembling Lung Tumor, with Case Report, G. F. Cooper, Ann. Int. Med., April, 1932, v, 1308.*—(*R. M. F.*)

Lung Abscess.—The anatomy and physiology of the lung is reviewed, and the relation to the development of abscesses is discussed. The most efficacious means to prevent abscesses are the employment of local anaesthesia in operations on the mouth and accessory sinuses, the utilization of such surgical technique as will minimize postoperative phlebitis, the prompt bronchoscopic removal of aspirated foreign bodies, and the earlier introduction of operative adjuncts in the treatment of pulmonary tuberculosis. Treatment for pulmonary abscess, particularly the gangrenous form, will be more effective if appropriate measures to promote adequate drainage are utilized as soon as posture alone is found to be inadequate.—*Pulmonary Abscess, J. L. Yates, Arch. Surg., August, 1932, xxv, 257.*—(*R. D.*)

Drainage of Lung Abscess.—In those cases of lung abscess in which the surrounding lung tissue is the seat of active, or increasing, purulent infection, internal drainage by extrapleural compression is indicated, in contradistinction to those cases of abscess where the surrounding lobules are clear, in which external drainage is the procedure of choice. The technique consists of resection of three or four ribs, leaving the periosteum of the removed ribs, over which dry gauze is packed, to produce pressure parallel to the bronchus draining the area, the course of the particular bronchus having been determined by previous studies. The packing is held by tight adhesive straps for 14 to 18 days, after which gauze packings are replaced every two days until rib regeneration has occurred, with the ribs in the compressed position. If this procedure is not satisfactory, partial thoracoplasties are done until the desired result is obtained, care being taken that the collapse is gradual. In one case a complete thoracoplasty was finally done. Of 14 patients treated by this method, 13 are recovering, and 9 are free from sputum. The one death occurred in a patient in whom internal drainage was not indicated since there was little surrounding pneumonitis, and external drainage, when finally performed, was too late. The essential purpose of this type of internal drainage is to eliminate the loss of the coughing reflex by which the purulent material surrounding the abscess can be evacuated; by external drainage the efficiency of this reflex is greatly diminished.—*Internal Drainage of Lung Abscess by Extrapleural Compression, F. S. Dolley, J. Thorac. Surg., April, 1932, i, 363.*—(*B. B.*)

Bronchoscopic Drainage in Pulmonary Suppuration.—The consensus of opinion is that an acute lung abscess failing to heal in two to three months should be referred for surgical intervention. If free drainage can be obtained in any way, the abscess will heal, and it is the possibility of obtaining free drainage by means of the bronchoscope that enables a cure to be made by this method. A series of 8 acute and 17 chronic lung abscesses treated by bronchoscopic drainage is reported. In acute cases treatment was given twice a week, and usually 5

treatments were sufficient to cause the abscess to heal. In chronic cases an average of 14 treatments at weekly intervals was necessary. *Results:* Of the 8 acute cases 7 were healed and one well on the way to recovery. This suggests that bronchoscopic drainage in acute cases produces better results than general medical treatment alone. Of 14 chronic cases 10 were healed, three were likely to heal, and one was referred to the surgeon. It is thus seen that in chronic lung abscess, bronchoscopic drainage offers more chance of success, with smaller mortality than surgical procedures, which in the past have produced 65 per cent cures and 35 per cent loss of life. In addition some of the surgical cures have been left with persistent sinuses very difficult to heal and often necessitating several plastic operations. In bronchiectasis bronchoscopic drainage is a palliative measure of very considerable value.—*The Bronchoscope in the Treatment of Pulmonary Suppuration, A. J. S. Pinchin and H. V. Morlock, Lancet, September 17, 1932, ccxxiii, 605.*—(*E. H. K.*)

Pneumonoconiosis.—Intensive studies of pneumonoconiosis have demonstrated the characteristic silica lesion. It is generally agreed that silica is soluble in the tissue fluids, and its action is chemical and not physical. The continued presence of crystalline silica in the lung is followed by the gradual development of a characteristic fibrosis. Asbestos produces a similar reaction but differs from it in the type and distribution of fibrosis. In regard to the action of silica in combination with other dusts, the author does not believe that the inhalation of silica combined with an alkali increases the solubility of the silica, nor that other dusts associated with silica protects the individual from the silica. There is abundant evidence that silica enhances the proliferative power of the tubercle bacillus. Although silica alone may produce such extensive pulmonary fibrosis as to cause death from cardiac failure, it is chiefly dreaded because of its association with tuberculosis. It is not merely a question of fibrosis, for there is evidence to show that silica combines with protoplasm and converts it into a particularly favorable pabulum for the growth of the bacillus. Infective silicosis is a slowly progressing lesion in which necrosis and fibrosis proceed side by side with a preponderance of the latter. It is believed that the tubercle bacillus is the chief infecting agent. Concerning the relationship of dusts to tuberculosis, silica and asbestos are the only known dusts which have any aetiological relationship. Inhaled dusts are dangerous only when they become soluble in the body and act as tissue poisons. Further experimental studies with various dusts are necessary before many problems of pneumonoconiosis are solved.—*Observations on the Pneumonoconioses, E. H. Kettle, Brit. M. J., August 13, 1932, no. 3736, 281.*—(*M. E. P.*)

Technique of Experimental Pneumonoconiosis.—The long period of time involved in producing experimental pneumonoconiosis has been a material handicap in the study of this condition. Pneumonoconiosis may be produced by the intratracheal injection of dust with the animal in the vertical position. One or 2 cc. of 3 or 4 per cent suspension in normal salt solution is used. The method is rapid, is attended by a relatively low loss of animals, and requires less dust, and much less care than dusting-chamber methods. A lung studied 14 days after injection showed no disorganization, but rather an appearance corresponding to that of dusted lungs. No matter how silica reaches the lungs time is an essential factor in the fibrotic process, and apparently the changes cannot be hastened.—*The Technique of Experimental Pneumonoconiosis, E. H. Kettle and R. Hilton, Lancet, June 4, 1932, ccxxii, 1190.*—(*R. D.*)

Pneumonoconiosis in Iron Foundrymen.—Parting compounds are used in iron foundries to dust the forms in order to separate the casts from the molds. Some of these compounds contain a high percentage of quartz and feldspar. One of the parting compounds in question contained a total of 81.5 per cent of silicic acid and silicates. The particles were small enough to enter the lower respiratory tract, the smallest ones measuring 0.75 micra. Four men working in a factory which manufactured parting compounds were all found to show roentgenological evidence of pneumonoconiosis. Their ages were 21, 23, 40 and 32. At the time of examination they had been in the factory for periods of 4, 3.5, 21 and 1 year. The latter had left the plant some time ago but roentgenological evidence of silicosis was convincing in his case. With the exception of the fact that 3 of the 4 workers showed evidence of pneumonoconiosis after relatively brief periods of exposure, the 4 cases are in no way different from cases of pneumonoconiosis in other industrial work.—*Formpuder als Ursache von Staublungenerkrankungen, A. Abraham, Klin. Wchnschr., June 25, 1932, xi, 1110.*—(*M. P.*)

Silicosis and Tuberculosis.—The X-ray picture is of great value in the diagnosis of pulmonary silicosis and has made possible a clinical division into stages of the disease, as in pulmonary tuberculosis. The dust nodules are formed earliest near the branching of the blood-vessels. Dust particles only below a certain size (10 mm.) may penetrate into the alveoli. These are responsible for the progression of the disease. It is not the mechanical so much as the chemical action (especially the solubility) of these particles which leads to pulmonary damage. Silicic acid (SiO_2) is not readily soluble but is chemically very active as it causes fibroblastic changes in the lung. It is long known that cavitation may develop in dust lungs. Postmortem examination of 6 cases of severe silicosis with cavity-formation revealed no evidence of tuberculosis in four. The wall of such a cavity is unlike a tuberculous cavity. There is no inflammation and no granulation tissue. The cavity results from liquefaction of the fibrotic tissue such as is seen in large myomata. That these fibrotic masses and nodules in silicotic lungs are not necessarily of tuberculous origin and not necessarily associated with a tuberculous process is illustrated in two cases of acute silicosis which came to autopsy. The formation of silicotic nodules and fibrotic tissue may occur without the presence of the tubercle bacillus. We should speak of a pulmonary silicosis with or without tuberculosis.—*Staublunge und Lungentuberkulose, W. Gerlach, Deutsche med. Wchnschr., February 19, 1932, lviii, 283.*—(*H. S. W.*)

Earliest Report on Asbestosis.—Since reports on asbestosis have been rather frequent during the last few years, the author points out that he reported a case as long ago as March, 1914, and that in this report he described the histological characteristics of the condition and the peculiar asbestos bodies.—*Kurze Bemerkungen zur Frage der Asbestose, T. Fahr, Klin. Wchnschr., June 25, 1932, xi, 1114.*—(*M. P.*)

Asbestosis.—Murray, in 1906, described a fatal case of pulmonary disease in an asbestos worker. At autopsy, extensive pulmonary fibrosis was found, which Murray attributed to the inhalation of asbestos dust. In 1924 Cooke reported a similar case complicated by pulmonary tuberculosis. He was the first to describe the curious bodies in the lung now known as "asbestosis bodies," while McDonald worked out the histological features. Later investigators showed that these bodies could be found in the juices expressed from affected lungs and also in the sputum. It is now known that the golden-yellow asbestosis body has a central core consisting of a minute asbestos fiber. In the present article two cases of pulmonary asbestosis are reported. *Case 1:* Of a white male who had worked as a spinner in an asbestos mill for nine years. Although the atmosphere was not very dusty he wore silver dust protectors in his nostrils. For five years following such employment he worked as a laborer. During the past five years he had morning cough, with moderate expectoration, some dyspnoea and palpitation. Chronic cholecystitis developed, a cholecystectomy was done, and the patient died of peritonitis. At postmortem, no gross pathological lesion of the lungs was demonstrable. On microscopical section, a few typical asbestosis bodies were seen in the alveolar walls. These bodies have a variety of appearances, but frequently are seen as rods or filaments with one end pointed and the other clubbed. The bodies are composed of a series of regular discs, very much as are red blood cells in *rouleaux* formation. They measure roughly from 10 to 200 micra and give the prussian-blue reaction for iron. There were scattered small collections of brown and blackish dust, free and within phagocytic cells, in fibrosed areas. Much of this dust and all of the asbestosis bodies stained positively for iron. The liver and the spleen were not pigmented. *Case 2:* Of an individual who suffered from both pulmonary tuberculosis and asbestosis. In 1921 he worked in an asbestos factory for about nine months. In the factory no precautions were taken against the inhalation of asbestos dust. In 1928, at the age of 52, he entered a hospital complaining of fatigue, loss of weight and a slight productive cough. Tubercle bacilli were present in the sputum at that time, and the roentgen examination showed a cavity in the right upper chest and a fine stippling in the lower lung fields suggesting pneumonoconiosis. The patient was discharged improved. Two years later the patient was admitted to the Jefferson Hospital for Diseases of the Chest, markedly emaciated and emphysematous. There were clubbing of the fingers and curving of the nails; the skin was slightly cyanotic. His course was progressively downward, bronchopneumonia developed, and death occurred approximately six months after admission. At no time during this period were tubercle bacilli found in the sputum. The roentgenogram showed small multiple cavity-formation in the right upper lobe, and the interlobar pleura below it was much thickened. In the lower lobes there was an increase in the markings resembling a change due to dust. At postmortem the heart

showed many firm, gray, translucent nodules on the free margins of the mitral-valve leaflets. The parietal pleura on both sides was thickly studded with small discrete, gray, pinhead-sized nodules, firm in consistency. Similar ones were present, much less frequently, on the visceral pleura. Over the bases of both lungs, firm, fibrous adhesions completely obliterated the pleural cavity. Between the bases of the lungs and the diaphragm, these adhesions were converted into a thick, tough, yellow, homogeneous substance having the appearance of hyaline cartilage. Both lungs were firm, the bases being particularly tough. The upper half of the right upper lobe showed a large multilocular cavity, the wall of which did not differ from the adjacent lung tissue. The cavity had a foul foetid odor, as of gangrene. Throughout the remainder of this lung were small grayish-red patchy areas. In the left lung were two round nodules, one measuring 4 cm. in diameter. It was firm and dense and composed of fibrous tissue, intermingled with gray cheesy material. The regional lymph nodes draining this area had a similar appearance. No tubercle bacilli were found in smears of the caseous areas. The nodules on the parietal pleura were tubercles of the productive type. Sections from the nodules in the left lung were examined for tubercle bacilli, but none found. The principal lesion of the lungs was a diffuse fibrosis, particularly marked in the lower lobes. The most striking feature in the lungs was the "asbestosis bodies." They were present in great numbers in the fibrosed areas, in some alveolar walls and spaces, and in the smaller bronchial branches. Particles of them were fragmented and were seen in phagocytic cells. In the caseous tuberculous areas in the left lung, these asbestosis bodies were fewer in number than in the adjacent lung tissue. In the spleen, the reticuloendothelial cells were loaded with a fine, amorphous, golden-brown and sometimes blackish dust. A few large typical asbestosis bodies were seen. Practically every hepatic cell contained a few granules of a golden-yellow pigment. In the kidney many cells filled with golden-brown granules were seen in the capillaries of the glomeruli. All bronchial nodes were fibrotic and pigmented. *Comment:* In the first case, as far as the patient's health and life were concerned, the presence of pulmonary asbestosis in the lungs was relatively unimportant. There is a possibility that the asbestosis was the cause of his emphysema and cough. It is to be noted that the patient was exposed for nine years in a comparatively well-ventilated factory in which precautionary appliances were used, and yet the patient developed a minor grade of the disease. Further precautions must be used in this industry. In the second case the patient worked in an asbestos factory for a short time, but without a mask. This was long enough to permit his lungs to become densely filled with dust. Obviously the symptoms did not result from the asbestos *per se*, but were due to the marked fibrosis resulting from the deposit of the asbestos dust. This demonstrates that the symptoms of a pneumonoconiosis may come on years after a short exposure and emphasizes again the absolute necessity for wearing masks in even the least dusty atmospheres. The marked fibrosis of the pleura, particularly at the diaphragm, is characteristic. The second case confirms previous observations that asbestosis bodies have a tendency to disappear in caseous areas. The finding of tubercle bacilli in the sputum (two different specimens), and the gross and microscopical features characteristic of tuberculosis, are sufficient to establish such a diagnosis. The asbestosis bodies were not looked for specifically in the sputum. An important finding in case 2 was that of asbestosis bodies in the spleen. Mention of such a finding is alto gether new.—*Asbestosis. Report of Two Cases, H. L. Stewart, C. J. Bucher and E. H. Coleman, Arch. Path., December, 1931, xii, 909.—(J. S. W.)*

Pulmonary Asbestosis.—Asbestosis of the lung is an occupational disease, a fibrosis of the lungs caused by irritation from silicic acid. Asbestos is magnesium silicate combined with potassium, aluminium and iron. Thirty-three employees who were exposed to asbestos dust have been under observation. They presented the following symptoms; cough, expectoration, paroxysms of dyspnoea, loss of appetite and weight, pleuritic pains and languor. Sputum as a rule was scarce, tenacious, mucoid and grayish. Four of the cases had a history of apical pulmonary tuberculosis and two of pleural effusion. In only 3 cases could tuberculous changes in the lungs be detected. In the X-ray picture tuberculous changes may be differentiated from those of asbestosis. In 10 of the cases percussion and auscultation of the lungs revealed no pathological changes. In the others, disseminated dry and moist râles were present. Early cases of pulmonary asbestosis show on the X-ray an increase in the pulmonary markings, associated with very small, disseminated nodules. The second stage shows numerous small, nodular, dense shadows, especially in the lower parts of the lungs. The third stage is characterized by

cloudy, soft, dense shadows. The demonstration of asbestos bodies in the sputum is of special diagnostic importance. These bodies give a deep blue reaction with potassium ferrocyanide and dilute hydrochloric acid. In asbestosis there is a marked discrepancy between clinical signs and subjective symptoms, and the X-ray picture. Even the most severe cases do not give such impressive X-ray pictures as those seen in silicosis. Asbestosis of the lungs may occur without preceding pulmonary tuberculosis.—*Ueber Asbestosis der Lungen, Gerbis and Ucke, Deutsche med. Wchnschr., February 19, 1932, lviii, 285.*—(*H. S. W.*)

Asbestosis Bodies.—The literature on this subject is reviewed and certain personal observations are added. A series of 44 forms of asbestosis bodies is depicted in an illustration. These bodies are apparently formed in the body by deposition of material about the asbestos fibre, the development of fissures in this material, and the separation of segments. The prussian-blue reaction of the bodies is probably not entirely due to the iron in the asbestos. Fibres were rendered iron-free by boiling with hydrochloric acid, then rendered alkaline and injected into the inguinal region of guinea pigs. A nodule in one animal, killed after 9 months, showed asbestosis bodies which gave the prussian-blue reaction for iron. This suggests that the iron is derived from the body tissues. Sputum containing asbestosis bodies, digested with artificial gastric juice for 24 hours, showed typical bodies persisting. When allowed to autolyze in an incubator for 14 days a very few imperfect bodies were found at the end of the period. Treatment by hydrochloric acid removes the golden yellow color but leaves a framework. Apparently the pigment and the framework represent different constituents. An attempt to produce the bodies artificially by exposing asbestos fibres to a number of reagents was unsuccessful.—*The Asbestosis Body, S. R. Gloyne, Lancet, June 25, 1932, ccxxii, 1351.*—(*R. D.*)

Adamantinoma of Jaw with Metastasis to Lung.—An adamantinoma is usually considered a benign tumor, but malignant transformation occurs. Metastases of these lesions are quite unusual. Following an operation at the age of 17 for a lump in the upper jaw, and five subsequent operations, a man of 36 had "dry pleurisy" and blood-streaked sputum. A year later a chest X-ray revealed a dense mass in the lower lobe of the right lung, and a dense nodule in the left lung. Despite X-ray therapy the patient died a year later. Autopsy revealed metastasis from the adamantinoma to the lung. The bronchi of the right lower lobe were markedly dilated and their lumina filled with a cast of the tumor tissue. In places, the lung parenchyma was invaded. It is suggested that the tumor tissue was aspirated from the primary tumor by way of the trachea and bronchial tree, and grew rapidly within the lumina of the bronchi. No similar metastatic lesion of an adamantinoma was found reported in the literature. —*An Instance of Adamantinoma of the Jaw with Metastasis to the Right Lung, J. Vorzimer and D. Perla, Am. J. Path., July, 1932, viii, 445.*—(*R. D.*)

Metastases to Lungs from Papillomata of Larynx.—A two-year-old girl had suffered from a dry cough and loss of voice for a year, and for two months had had occasional marked nocturnal dyspnoea. By direct laryngoscopy numerous papillomatous masses were removed from the larynx. On section these masses showed benign papillomata. During the next two years the patient was treated ten times, radium being inserted on two occasions, a tracheotomy tube being necessary during the last year. At autopsy the whole of the larynx and the upper part of the trachea were found to be filled with cauliflower-like masses of papillary structure. These were grayish-white and firm. In the lungs there were many small cavities whose connection with the smaller bronchi could be traced. The walls of these cavities were covered with fine granules about the size of millet seeds. Careful study of microscopical sections suggests that these secondary foci were due to the detachment of masses from the laryngeal growth and their aspiration into the bronchioli where implantation occurred. No invasion of lymphatics was observed.—*A Case of Multiple Papillomata of the Larynx with Aerial Metastases to Lungs, H. B. Hitz and E. Oesterlin, Am. J. Path., May, 1932, 333.*—(*R. D.*)

Roentgenology of Primary Cancer of Lung.—Roentgenological analysis of 36 cases of primary carcinoma of the lung was made. It revealed that 8 were in the right upper lobe, 5 in the right middle lobe, 8 in the right lower lobe, and 9 in the left lower lobe. Most of these also involved the root structures. Of those involving the root structures, 9 were on the left side and 15 on the right. The outline was sharp in 23 cases and irregular in thirteen. Twenty-six showed some evidence of displacement of the heart and other mediastinal structures toward the side of the lesion; 3 showed evidence of displacement to the side away

from the lesion; 7 cases showed practically no displacement. In 21 cases the area was large, in 9 cases medium, and in 6 small. Metastases were evident in 6 cases. There was bronchoscopic evidence of new growth in 29 cases, surgical biopsy confirmation in 3 and postmortem confirmation in five. The pleura was involved in 6 cases and not involved in thirty. Definite difficulty lies in distinguishing between primary carcinoma that presents a sharply defined tumor mass in contact with the mediastinum and new growth that may arise from the mediastinum. Lesions, such as Hodgkin's disease or others of the lymphoblastoma-type tumors of the thyroid and thymus, must be considered. With mediastinal new growths or lymph-node enlargement, the trachea, aorta, and so forth, are usually displaced away from the lesion. Twenty-one of these cases received roentgen treatment. All of these treated cases have made at least temporary clinical improvement. One patient is living and well, except for a permanent bronchiectasis, for seven years; another six; another nearly eight years; and a number have gone symptom-free for more than one year. A few of the advanced cases have shown only temporary improvement and then rapid progression.—*Primary Carcinoma of the Lung: Roentgen Diagnosis and Preliminary Report on Roentgen Therapy, W. F. Manges, Am. J. Roentgenol. and Rad. Ther., June, 1932, xxvii, 858.*—(*W. I. W.*)

Roentgenology of Cancer of Lung.—The relatively rare, spherical shadows in the X-ray picture of the thorax present difficult differential diagnostic problems, as they may indicate dermoid cysts, echinococcus cysts, primary carcinoma and sarcoma, abscess, pleural exudates, infarcts, or actinomycotic disease. In this case physical examination showed moist râles in the upper half of the chest. The X-ray revealed old, bilateral, apical tuberculous foci. In the right upper lobe there was a sharply defined shadow, the size of a dollar, which increased in two months to the size of a man's fist. The diagnosis of carcinoma was made at the autopsy five months after the apparent onset of the disease. This occurred in a man of 79 years with a previous history of tuberculous epididymitis and spondylitis.—*Das zentrale Lungenkarzinom als Rundschatten im Roentgenbild, J. Leeser, Deutsche med. Wchnschr., July 8, 1932, lviii, 1089.*—(*H. S. W.*)

Tubercle in Hodgkin's Disease.—The exciting cause and the identity of the important pathologicoanatomical structure of the clinical syndrome described by Hodgkin in 1832 remain in doubt. The controversy has narrowed itself down to a question of infection, malignancy and haematopoietic hyperplasia. Many observers have reported tubercle bacilli present in the lesions of Hodgkin's disease, and some investigators consider this disease a "peculiar form of tuberculosis" (Sternberg). Although negative reports are many, it is now generally conceded that tubercle bacilli may be present in the lesions, but as secondary invaders. The lesion of Hodgkin's disease may afford favorable soil for the growth of these bacilli (Weber). Those who have tried to demonstrate by means of the tuberculin reaction that Hodgkin's disease springs from tuberculosis have obtained only negative results. The present article deals with a case of advanced Hodgkin's disease in which it was possible to demonstrate one of the earliest primary tuberculosis complexes yet recorded. A six-year-old white girl died of Hodgkin's disease after an illness of fourteen months. There was no history of contact with tuberculosis, and the sputum and urine were always negative for tubercle bacilli. At postmortem the following abnormalities were observed: Small, early, primary tuberculous lesion at the apex of the right lower lobe, with a lymphatic complex by way of the bronchial and pleural lymphatic vessels; bronchopneumonia of both lower lobes; Hodgkin's disease of the cervical, periaortic, peripancreatic and peribiliary lymph nodes, spleen and liver, marked anaemia, and other minor conditions. The pulmonary tuberculous lesion, situated just beneath the pleura, measured 3 mm. across the caseous portion, with a ring of inflammatory tissue from 2 to 3 mm. wide around it. There were a few smaller foci around the larger one, but they did not show caseation. Microscopically, the principal lesion revealed a slight excavation in what appeared to be a terminal bronchiole with a slight zone of caseation around it undergoing sloughing. The main body of the lesion consisted of early caseation of the bronchopneumonic focus, which is considered specific for a primary lesion by Ranke, Pagel and Henke and others. From this local site the infection traveled toward the hilum by two routes: (1) the pleural lymphatic vessels and (2) the bronchial lymphatic vessels. Microscopically these lesions showed a typical infiltration with Langhans giant cells, "epithelioid" cells and all the other characteristics of a tuberculous process. No atypical cells could be found. No Sternberg-Reed cells were present in the lungs, pleura or their lymphatic vessels. The lesions in the cervical and peripancreatic lymph nodes and the

lesions in the spleen and liver were typical of Hodgkin's disease. There was no process that could be mistaken for tuberculous inflammation, although tubercle bacilli were present on inoculation into animals. The diseased lymph nodes and splenic nodules were macerated separately and each prepared for a series of inoculations. Tissues from guinea pigs developing tuberculosis as a result of these inoculations were reinoculated into guinea pigs, chickens and rabbits, and only the first developed tuberculosis. Berkefeld filtrates of macerated nodules from the spleen and lymph nodes were inoculated into guinea pigs and chickens with negative results. It was concluded, therefore, that only human tubercle bacilli were present in the patient, and that no filterable forms could be demonstrated. There seems to be clear evidence that a primary tuberculous infection occurred in the lungs of this patient fully a year after the Hodgkin's disease had developed, for, from the point of view of age, the tuberculous lesion appeared to be similar to those in animals at from four to six weeks after infection. The finding of tubercle bacilli in the abdominal lymph nodes that showed Hodgkin's disease can easily be explained. The haematogenous phase of a primary lesion the size of this one may not cause extrapulmonary metastases, although bacilli certainly are disseminated in varying numbers from the focus. There is no proof that tuberculous infection was present except in the pulmonary focus. The only question is whether this focus was primary or secondary. The main point of difference between a primary and a secondary tubercle is that in the former the caseous bronchopneumonia evolves much like an ordinary pneumonia except that it goes on to caseation, calcification and sometimes excavation, but there always remains the basic tissue framework (in the unexcavated part) which may be demonstrated by simple staining. Weigert's stain of a cross-section of this tubercle showed a complete framework of the alveoli and blood-vessels up to the edge of excavation. Around the periphery the alveoli were plugged with fibrin containing scattered polymorphonuclears, lymphocytes and "epithelioid" cells. There were many plugs of almost pure fibrin. Epithelioid cells were numerous in certain places, but there was no sign of a cellular or fibrous capsule. In contrast, a secondary tubercle has an avascular centre with homogeneous, obliterative caseation necrosis or a secondary bronchopneumonia that is diffuse, extensive and irregular, with no signs of encapsulation and little or no lymph-node complex. The isolated lesion described, seems, therefore, to be primary. Although this does not disprove the theory that Hodgkin's disease, in other instances, is infectious in origin, it practically rules out tuberculosis as a cause in this case, and with it the foundation stones of the whole infection theory are severely shaken. Attention is called to the view that Hodgkin's disease may be a malignant process. As most malignant diseases are initiated by an irritant, it is not an unreasonable theory to suppose that the variety of agents found represent at most a form of irritation that is of such a low grade of virulence that only a toxic reaction is produced without any constant specific changes. It seems justifiable to conclude that Hodgkin's disease in the patient studied developed independently of tuberculous infection, and that the theory of a tuberculous origin of the disease is not supported by this case.—*A Primary Pulmonary Tubercle Appearing in a Patient with Advanced Hodgkin's Disease, H. C. Sweany, Arch. Path., April, 1932, xiii, 558.*—*(J. S. W.)*

Cancer Following Bone Tuberculosis.—The history is given of a patient who, at the age of three and after trauma, had developed a tumor on the left hip with a discharging sinus. On admission a cold abscess and extensive tuberculosis of the left hip were found. Six months later, after treatment with a cauterizing ointment, an epithelial-cell carcinoma was diagnosed and verified by biopsy. The case resembles that reported by Hoche, in which X-ray treatment was applied. The question is raised, whether the use of X-rays or the application of a cauterizing ointment are contributory aetiological factors in the development of a carcinoma on an old tuberculous soil.—*Ueber die Vergesellschaftung von Tuberkulose und Karzinom.—K. Rummelbach, Wien. klin. Wchnschr., August 19, 1932, xlv, 1040.*—*(K. F.)*

Tuberculin in Hodgkin's and Other Diseases.—For many years much work has been done on the question of the relation of tuberculosis and Hodgkin's disease. Some investigators have concluded that Hodgkin's disease is an atypical form of tuberculosis; others that it is caused by some peculiar type of tubercle bacillus; and still others that there is no connection between the two diseases. The problem has been attacked bacteriologically and by means of the tuberculin and precipitin reactions. The present paper is concerned with the tuberculin reaction of patients with Hodgkin's disease and other diseases of the lymphoid and myeloid tissues. Apparently, the great majority of cases of Hodgkin's

disease give a negative skin reaction to tuberculin, and in addition the reaction is often negative even in cases with an associated active tuberculosis. The reason for this anergy is not evident and does not seem to depend on the stage of the disease. The present study has two purposes; first, to compare the tuberculin reaction of patients with Hodgkin's disease with reactions in diseases of the lymphoid and myeloid tissues; second, in view of L'Esperance's claims as to the aetiological importance of avian tuberculosis in Hodgkin's disease, to test the reaction of cases of this disease to avian tuberculin as well as human. Avian and human tuberculins were prepared in an identical manner except that the reaction of the glycerin-broth in which the avian strain was grown was slightly alkaline. The avian tubercle bacilli grew luxuriantly throughout the broth in a characteristic fashion, whereas the human tubercle bacilli formed a typical pellicle on the surface. No mention is made of the source of either strain. After six weeks' incubation, tuberculin was prepared from both strains in the usual way. The tuberculins were tested out on two small groups of guinea pigs, each group having been inoculated four weeks previously with the homologous organism. Normal guinea pigs gave no reaction (intracutaneous) to either tuberculin, whereas the inoculated animals tended to react more strongly to the homologous tuberculin. Each patient was injected intracutaneously, first, with 0.1 cc. of a 1:10,000 dilution of each tuberculin. The results of these injections were read forty-eight hours later. If either or both were positive, no further tests were made. If negative, the patient was retested with 0.1 cc. of a 1:1000 dilution. Again, if there was a positive reaction, no further tests were made. The patients utilized in this study included 16 cases of Hodgkin's disease, 15 cases of other types of malignant lymphoma, 6 cases of leukaemia, 16 cases of pernicious anaemia, 23 cases of malignant tumor, 27 cases of active tuberculosis, and, as controls, 21 normal individuals. A greater number of patients reacted to the avian than to the human tuberculin with the exception of the normal individuals. Fewer positive reactions were obtained in the patients with diseases of the lymphoid and myeloid tissues and with malignant disease than in normal and tuberculous patients. Furthermore, none of the patients with disease of the lymphoid-myeloid tissues gave a positive reaction to human and a negative to avian tuberculin, while the reverse was true in a considerable number of instances. The Hodgkin's disease and tuberculosis groups gave the highest number negative-to-human-positive-to-avian reactions, 15.4 per cent and 13.3 per cent, respectively, with the 1:1000 dilution of tuberculin. The authors have no explanation to offer as to why there should have been more positive reactors to the avian tuberculin. Why the groups other than the tuberculous and normal gave a much lower percentage of positives is also unknown. The patients with malignant disease and pernicious anaemia were all hospitalized and the majority were debilitated and such debility might be considered responsible for the reduced number of positive reactors in these two groups. However, this explanation could not be applied to the Hodgkin's disease and malignant lymphoma patients, as more than 75 per cent of them were doing their daily work and only two were hospitalized. If Hodgkin's disease is due to tuberculosis, it would be reasonable to expect that the number of positive tuberculin reactions would closely approach that of patients with tuberculosis alone. However, the anergy produced by Hodgkin's disease would tend to offset any such tendency to increased reactions. It would seem, therefore, that the tuberculin test as an indication of the relation of tuberculosis to Hodgkin's disease is of doubtful value.—*Studies of Diseases of Lymphoid and Myeloid Tissues. IV. Skin Reactions to Human and Avian Tuberculin, F. Parker, Jr., H. Jackson, Jr., G. F. Hugh and T. D. Spies, J. Immunol., April, 1932, xxii, 277.*—(*J. S. W.*)

Pyopneumothorax in Hodgkin's Disease.—A woman of 23 was admitted to the hospital after an indefinite illness of 12 weeks' duration which had been marked by pain in the back and irregular fever. Two weeks before admission severe stabbing pain was noted in the right chest, and this was made worse by coughing or taking a deep breath. A thoracocentesis revealed no fluid. An X-ray taken a few days after admission showed a pocket of air in the costophrenic angle on the right. The pneumothorax increased, and a few days later pus, containing streptococci, was found in the right chest. Death followed the development of peritonitis. The autopsy showed a right pyopneumothorax, but no obvious communication with the lung. There was a nodule in the right main bronchus, the hilum lymph nodes were enlarged, a tumor mass was present in the liver, and the retroperitoneal nodes were enlarged. The diagnosis was Hodgkin's disease. The pneumothorax was an accidental complication which had obscured the underlying condition. It is of note that physical examination had revealed no en-

largement of the lymph nodes and the spleen was not palpable. The Hodgkin's disease evidently originated in the deep seated lymph nodes.—*Pyopneumothorax in Hodgkin's Disease, A. J. Hall and R. Y. Dawbarn, Lancet, January 23, 1932, ccxxii, 183.*—(*R. D.*)

Syphilis of Lung.—A male, aged 42, was hospitalized because of haemoptysis. The only other symptoms were slight weakness and a productive cough of two months' duration. The family and past histories were negative except that acute rheumatic fever had occurred nineteen years previously, and heart disease, which did not incapacitate the patient, was diagnosed about twelve years before admission. Physical and roentgenographic examinations revealed decreased aëration and increased density in the lower half of the left hemithorax, with slight displacement of the heart to the right. Seventeen days after admission a large effusion developed rapidly in the left pleural space. Large amounts of serosanguineous fluid were aspirated for relief of dyspnoea. Nonproductive cough persisted. Weakness, dyspnoea and cyanosis increased, haemoptysis occurred, and the patient died. The sputum was negative for tubercle bacilli on 14 occasions and also upon inoculation of a guinea pig. The blood Wassermann was strongly positive. The chief findings at necropsy were as follows: Abnormal amounts of serous fluid were found in the pericardial, peritoneal and left pleural spaces. In addition, about 400 cc. of pus were found between the base of the left lung and the diaphragm. Posterolateral to the bifurcation of the bronchi on the left was found a circumscribed mass of 10 cc. of pus. In apposition to the upper portion of the lower left lung was found a sacculated aneurysm of the descending aorta. A seeping haemorrhagic tract led from it into the substance of the lower posterior portion of the left lung. The right lung was heavy and dark gray, and the pleura was thickened. Both lungs, on section, were found to contain diffuse tubercles, and in the upper lobes small cavities and caseous material were found. In the lower lobes of both lungs there was carnification, and in the lower left lung an increased amount of the stroma without aëration. Near the base of the left lung was a gray rubber-like mass the size of a five-cent piece, which was diagnosed as a gumma. The tubercles were considered to be gummata. There was no evidence of tuberculosis or of neoplasm. There was a chronic epicarditis; also focal areas of interstitial myocarditis. The complete anatomical diagnosis was as follows: Syphilitic aortitis; aneurysm of the descending aorta; chronic fibrinous pleurisy; empyema, encapsulated, left side; pneumonia, lobar type, gray stage; serofibrinous pleurisy of the right side with atelectasis of the lower right lung; atelectasis of the lower left lung; chronic passive congestion of the liver and spleen; acute nephrosis with vascular nephritis; hydropericardium; gumma of the left lung. A review of the literature on syphilis of the lung is appended. Apparently there is no distinctive finding characteristic of this condition. The pathological findings may be indistinguishable from those of tuberculosis. Schmorl found spirochaetes indistinguishable from *Spirochaeta pallida* in pulmonary gangrene and aspiration pneumonia.—*Syphilis of the Lung. Report of a Case with Autopsy Findings, H. C. Denman, Ann. Int. Med., January, 1932, v, 895.*—(*R. M. F.*)

Diabetes and Tuberculosis.—Though the combination of diabetes with pulmonary tuberculosis has been studied for many years, the opinions as to diagnosis, course, prognosis and treatment are still divided. This report is based on 70 cases of diabetes in which pulmonary tuberculosis was diagnosed. The incidence of pulmonary tuberculosis in all diabetes patients seen between 1925 and 1931 was only 7 per cent against a much higher percentage found by other observations, and seems to have decreased with the use of insulin. The cause of the frequency of phthisis and diabetes is still a matter of conjecture; the assumption that the hyperglycaemic blood is a good culture medium for tubercle bacilli or other microbes has been repeatedly refuted and the lowered resistance of the diabetic to infections can be better explained by the disturbance in the carbohydrate metabolism, because in insulin-treated patients the pulmonary disease takes a more typical course than in the untreated cases. In the latter group the fever is much lower than would be expected with an extensive destructive process, nightsweats are extremely rare or absent, and the sputum is only slightly positive and is raised in surprisingly small amounts. With the use of insulin all symptoms begin to resemble more those seen in the nondiabetic tuberculous. The localization and the type of phthisis mostly found in diabetes is as atypical as the symptomatology. In 15 cases, the middle portion was involved (21.4 per cent) while the apices were entirely free. The great majority in this group had exudative forms. In 32 cases (42.8 per cent) a fibrocaseous type was found, while in 18 instances (28 per cent) the process was fibroulcerative in character. It was found

that the extent and the character of the pulmonary involvement was not closely related to the degree of diabetes, and even very severe cases of diabetes showed a benign fibrous type of pulmonary tuberculosis. The diagnosis in the treated cases does not present many difficulties because the course of the pulmonary disease becomes more typical and many pathognomonic symptoms point definitely to phthisis. It is pointed out that the physical findings in the treated cases correspond with the amount of pathological involvement and that the tuberculin tests were negative in only 16 per cent while the sedimentation rate was markedly increased. The course of the pulmonary disease depends on the way the diabetes reponds to diet and insulin. If it is possible to keep the patient permanently sugar-free, his chances for improvement are very little affected by his diabetes. The more progressive course which was noted in some cases is explained by economic factors. While the treated diabetes is not to be considered a serious complication, the authors observe that the coexisting pulmonary tuberculosis usually decreases the sugar-tolerance of the diabetic. The prognosis of the diabetic tuberculous is not as unfavorable as was formerly thought, provided the diabetes can be kept under control. It seems for this reason to be advisable to treat at once the diabetes with insulin and not to lose too much time in attempting to render the patient sugar-free by diet alone. In no instances were untoward effects of insulin injections, such as mild focal reactions which have been described by other writers, observed, but hypoglycaemic shocks which are often followed by severe and profuse perspiration have to be avoided. The importance of proper diet is, of course, stressed. All patients in this group were at first given Falta's standard diabetic diet, which consists of approximately 150 gm. of meat, two eggs, coffee, 400 to 600 gm. of vegetables, 150 gm. of fat, 200 gm. of cream, 30 gm. of rice, 120 gm. of potatoes, and 40 gm. of white bread. As soon as the sugar tolerance has been tested, the diet has to be individualized, according to the requirements of the case, to the units of insulin which are needed and to the condition of the lung. The patient should not be discharged from the hospital before his lung condition is stationary and until his nutrition has been so improved that his weight is normal for his age and height. In order to continue the insulin treatment on ambulatory patients, permanent and regular control and the cooperation of the patient are indispensable.—*Klinische Erfahrungen an tuberculösen Diabetikern, die mit Insulin behandelt wurden, G. Delijannis and G. Petassis, Wien. klin. Wchnschr., July 15, 1932, xlv, 909.*—(*K. F.*)

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Calcium Deficiencies IN TUBERCULOSIS

IN a study of 154 tuberculous patients Kaminsky and Davidson[1] found low calcium values to be more frequent than in normal subjects. Crimm suggests that a slight hypercalcemia may be desirable in the treatment of tuberculosis and employs viosterol for the purpose.[2]

Spies[3] and Levaditi and Po[4] found in experiments that viosterol has the special property of calcifying the necrotic and caseous portions of the tubercles. The latter state that a calcium shell is formed, probably the bacteria-inhibiting factor.

Hildebrand[5] notes that where the incidence of rickets is high, that of tuberculosis is likewise. Grant, et al.,[6] have shown ricketic rats to be more susceptible to tuberculosis than are non-ricketic animals. Bergmann[7] finds that both rickets and tuberculosis respond to treatment with irradiated ergosterol. Menschel speaks highly of viosterol for tuberculosis, stating that it inhibits the exudative processes and also checks nightsweats, fever, and hemorrhages.[8]

Present-day thought on calcium therapy in tuberculosis is well summarized by the following statement in the *Journal of the American Medical Association:*[9]

"In previous years certain chemical substances—principally the creosote series—were used to inhibit the growth, or to destroy, tubercle bacilli in the body. Tuberculosis workers have learned the inadequacy of such measures and these substances have been discarded almost entirely. It is felt that the improvement in the patient, consequent on the taking of a balanced dietary, with an adequate amount of mineral salts and vitamins, allows of an increased phagocytosis, which increases the destruction of tubercle bacilli. The improved condition of the tissues also enables not only a resorption of the inflammatory exudate in tuberculosis but a walling off of the tuberculous process, thereby preventing the spread of such disease. Calcium therapy in tuberculosis is considered one of the most important considerations. Calcium absorption is apparently dependent on the vitamin D function in the body."

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[1-10] *Bibliography on request.*

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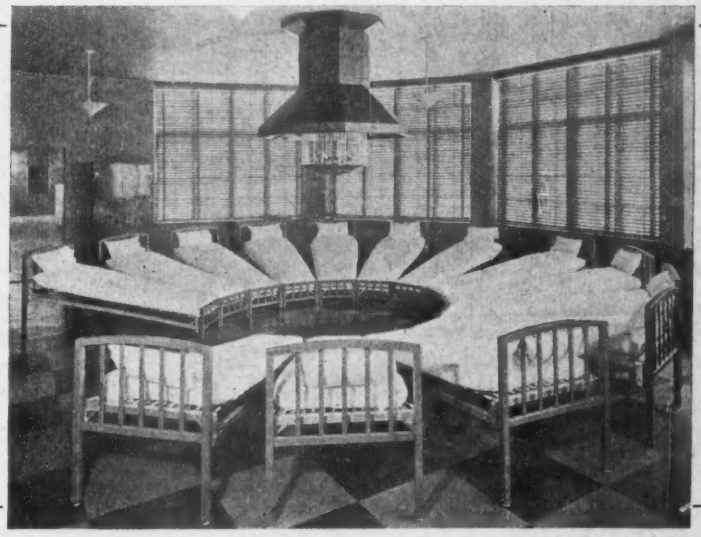

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Further, it has been shown that there exists a definite balance between vitamins; by greatly increasing vitamins A and D in diets, an otherwise adequate amount of vitamin B is made inadequate—leading to death of animals. Care should be taken not to overdose with cod liver oil or viosterol unless, at the same time, an increasing quantity of vitamins B and G are also added to the diet. As a result of new information, there is a growing tendency on the part of the medical profession to advocate the administration of vitamins in group form.

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